

AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph at page 4, lines 2-8, as follows:

An embodiment of the medicine is that the angiotensin-converting enzyme is a mutant angiotensin-converting enzyme to which one or more amino acid mutation is introduced so that GPI-anchored protein releasing activity is maintained but peptidase activity is inactivated. Preferably, the mutant enzyme has one or more amino acid substitution in the sequence of His Glu Met Gly (the 413th to 416th amino acid residue of SEQ ID NO: 4), and most preferably, the mutant enzyme has Glu to Asp amino acid substitution in the sequence of His Glu Met Gly His (the 413th to 417th amino acid residue of SEQ ID NO: 4).

Please replace the paragraph at page 4, lines 14-16, as follows:

The mutant enzyme has one or more amino acid substitution, and preferably has Glu to Asp amino acid substitution in the sequence of His Glu Met Gly His (the 413th to 417th amino acid residue of SEQ ID NO: 4).

Please replace the paragraph at page 12, line 22 to page 13, line 3, as follows:

Although it would be possible to accordingly design the mutation introducing sites for preparing the mutant ACE, the present invention provide as a preferable one the mutant ACE that carries more than one amino acid substitutions in the amino acid sequence of His Glu Met Gly His (the 413rd-417th of SEQ ID NO: 4) in the ACE amino acid sequence. This sequence region consists zinc binding site necessary for peptidase activity and are almost completely conserved among the reported ACE, either ACE-T or ACE-S, from all species, such as mammalian (human, mouse, rat, rabbit, cow), avian (chicken) and insect (fly). The present invention further provides, as a more preferable one, a mutant ACE having Glu to Asp substitution in the sequence of His Glu Met Gly His (the 413th to 417th amino acid residue of SEQ ID NO: 4) (hereinafter, the mutant may be referred referred to as “peptidase inactivated ACE (E414D)”).

Please replace the paragraph at page22, lines 20-28, as follows:

The effect of ACE activity was further examined for CD59 and the decay-accelerating factor (DAF) in HeLa cells; and prion protein in HEK293 cells by using FACS analysis (Figures 10 and 11). As a result, it was confirmed that all proteins were efficiently shed from cell surface. ACE shedding activity for CD59 in HeLa cells was more clearly in the case of disrupting lipid raft by treatment of cholesterol blocking agent, filipin (Figure 10), which effect was ACE dose-dependent (Figure 11_12) and was inhibited by captopril (Figure 12_13). In contrast to F9 cell molecules, GPI-anchored proteins on human cells were readily released from the cell surface without filipin treatment (Figure 14).